Letters to the Editor

Re: Postoperative ventilatory dependency following thymectomy for myasthenia gravis

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Editor.—

We read with interest the paper by Sivak et al. Our experience involving the need for postoperative ventilation is very different from that described by these authors. In our series of 29 trans-sternal thymectomies, only 2 patients required postoperative ventilatory support. Also, no information was given by the authors about either the postoperative analgesia regimen or the general anesthetic technique, both of which could contribute to the need for postoperative ventilation.

Our 29 trans-sternal thymectomies (20 for myasthenia gravis and nine for thymomatous myasthenia) were performed over a period of 50 months. The patients were similar to those described by Sivak et al with regard to age, sex, and duration of symptoms. The Osserman classifications for our patients were stage I (7%), stage IIA (48%), and stage IIB (45%). Although there is a difference in the disease staging between our patients and those of Sivak et al, there is no correlation between their patients' Osserman classification and the requirement for postoperative ventilation.

Our anesthetic technique consisted of

- 1. Premedication. The premedication included papaveretum (10-20 mgm) and hyoscine (0.2-0.4 mg, administered one hour postoperatively). Dosages were determined per 1.7 m² body surface area.
 - 2. Induction. Thiopentone (250-350 mg, ad-

ministered intravenously) and succinylcholine (100 mg) were given. Also, 4% lignocaine spray was applied to the larynx and pharynx prior to oro-endotracheal intubation.

- 3. Maintenance of anesthesia. Intermittent positive-pressure ventilation was maintained with 66% nitrogen in oxygen with 0.5%-1% halothane (volume, 7-10 L/min; frequency, 12/min). At the end of the operation, atropine (1.2 mg) and neostigmine (2.5 mg, administered intravenously) were given. All patients were extubated following clinical assessment of level of consciousness, ventilatory adequacy, and evaluation of muscle power by the ability to raise the head from the pillow for five seconds on command.
- 4. Postoperative analgesia. Intermittent papaveretum (10-15 mg) was administered intramuscularly three to four hourly, as required.
- 5. Drug management. Twenty-five patients were taking anticholinesterases (pyridostigmine) orally preoperatively. No anticholinergic therapy was given prior to surgery on the day of the operation. Neostigmine was given at the end of the operation, and oral anticholinesterases were restarted at the preoperative dosage two hours after surgery. Seven patients were taking prednisolone preoperatively. This was restarted on the day following the operation at the preoperative dosage levels.

Only 2 of the 29 patients required ventilation for longer than 24 hours. One patient was a 60-year-old man with nonthymomatous myasthenia who presented with stage IIB disease. He had a lower respiratory tract infection preoperatively, but his debilitating myasthenia warranted an early thymectomy. Sputum retention necessitated ventilation for 48 hours postoperatively. The other patient was a 48-year-old man who had thymomatous myasthenia (stage IIA). A large thymoma was found and resection of this tumor entailed the loss of the left phrenic nerve. Postoperatively, left-sided diaphragmatic palsy developed and the patient required ventilation for 30 hours.

Our analysis of the paper by Sivak et al reveals evidence supporting the immediate commencement of oral anticholinesterases postoperatively. Fourteen of their 17 patients requiring ventilation for less than 48 hours were given anticholinesterases within 24 hours postoperatively, whereas only 2 of the 7 patients requiring ventilation for longer than 48 hours re-

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ceived anticholinesterases within 24 hours postoperatively. We agree with their summary that following thymectomy prompt institution of cholinesterase inhibitor therapy is necessary to facilitate the weaning from mechanical ventilation. However, we believe that postoperative mechanical ventilation can be avoided in most patients (93% in our study) with our current anesthetic technique combined with immediate resumption of administration of preoperative anticholinesterase agents. Postoperative mechanical ventilation is only required when there is a complication from the surgery or a chest infection.

Myasthenia gravis is a disease caused by circulating antibodies originating from the thymus.² Therefore, it is reasonable to postulate that effects on the thymus will, in turn, increase the level of maintained circulating antibodies and that perhaps anticholinesterase administration should continue during the operation to help preserve neuromuscular junction function.

We were intrigued to note that in the work by Sivak et al, pain from chest drains and median sternotomy prolonged the need for ventilation in 3 patients and that 1 patient required ventilation following sedation for sleep deprivation. Similar problems did not occur in our study.

Several other parts of their paper are puzzling. In Table 1, patient 10 is classified as stage IIIB—which does not exist in the Osserman classification.³ In Table 3, patient 14 appears to be taking two separate doses of steroids; one of these should presumably be pyridostigmine. Also, patient 22 underwent a resection of the left phrenic nerve during surgery, but appeared to suffer postoperatively with a bilateral diaphragmatic palsy. Can all this be explained?

References

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Dr. Sivak et al reply.—

Doctors Graham, Pearson, and Holden present observations that reinforce our recommendations that immediate postoperative antiocholinesterase therapy following thymectomy will facilitate weaning from mechanical ventilation. Although the numbers and types of patients in our series and the study of Graham et al are similar, one distinct difference is that our

patients did not receive any therapy until 24 hours postoperatively (i.e., the first postoperative day). This point should be emphasized since *Table 2* indicates that medications were started on day 1 (the day after surgery) or after.

Another difference is that our weaning criterion was based on a patient's ability to maintain ventilation as mechanical support was gradually decreased. Dr. Graham et al used a general assessment of muscle strength which correlated with the ability to support minute ventilation. Our weaning process may have been shortened considerably if this criterion had been used.

Patients 2, 3, 13, 14, 19, 21, 22, and 24 received no preoperative medications. Patients 1, 4–7, 9, 10, 18, and 23 received hydroxyzine pamoate (50 mg) and meperidine hydrochloride (50 mg). Patients 8, 11, and 17 were given promethazine hydrochloride 25 (50 and 15 mg, respectively). Patients 12 and 15 received morphine sulfate (10 mg). Patients 2 and 7 were given diphenhydramine hydrochloride (50 mg).

Induction was done with thiopental sodium (1.8 to 11 mg/kg). Maintenance anesthesia was appropriate mixtures of nitrous oxide, oxygen, and either halothane (15 patients) or enflurane (9 patients). Atropine and neostigmine were not administered after surgery.

We found no group difference or similarities when comparing preoperative medications or anesthetic agents.

Graham et al also noted some minor errors. Patient 10 had stage III disease, and patient 14 was taking pyridostigmine (60 mg daily) and prednisone (15 mg daily) preoperatively.

We are unable to explain the bilateral diaphragmatic paralysis in patient 22. This problem, of course, can occur after heart surgery and suggests some common factor such as stretch or heat injury from electrocautery.

We thank Dr. Graham and his colleagues for providing a series of patients for comparison to our own. Their observations have helped us meet our original goal to identify factors that might prolong ventilatory support in the postoperative period.

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